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A STUDY ON NON TRAUMATIC ULCER IN UPPER LIMB AT GRH, MADURAI.

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CERTIFICATE

This is to certify that the dissertation entitled, **"STUDY ON NON TRAUMATIC ULCER IN UPPER LIMB" AT GRH, MADURAI"** submitted by **Dr.K.HEMALATHA** to the Tamil Nadu Dr.M.G.R. Medical University, Chennai in partial fulfillment of the requirement for the award of M.S. Degree Branch-I (General Surgery) is a bonafide research work were carried out by her under direct supervision & guidance

DR.V.Seetharaman, M.S.,
Unit Chief
Govt. Rajaji Hospital
Madurai Medical College
Madurai

Dr.M.Gobinath, M.S.,
Professor and HOD,
Govt. Rajaji Hospital,
Madurai Medical College
Madurai.

DEAN
MADURAI MEDICAL COLLEGE
MADURAI.

DECLARATION

I **Dr.K.HEMALATHA** declare that, I carried out this work on, **"STUDY ON NON TRAUMATIC ULCER IN UPPER LIMB" AT GRH, MADURAI,** at the Department of surgery, Govt. Rajaji Hospital during the period of June 2006 to June 2008. I also declare that this bonafide work or a part of this work was not submitted by me or any others for any award, degree, diploma to any other University, Board either in India or abroad.

This is submitted to The Tamilnadu Dr.M.G.R. Medical University, Chennai in partial fulfillment of the rules and regulations for the M.S. degree examination in General Surgery.

Place: Madurai. **Dr.K.HEMALATHA**

Date:

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CONTENTS

INTRODUCTION

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The ulcers of upper limbs constitute one of the common surgical problem with varied etiology, disease manifestation and treatment.

The concept of the whole management rested on clinical examination and differential diagnosis has changed. The newer modalities of investigations like ultra sonogram, computed tomographic scan, Arteriography and magnetic resonance angiography have revolutionized the approach to the ulcer of the upper limb.

Peripheral vascular disease is a serious condition that increases the individual and population based risk of heart attack, stroke, death and amputation and decreases the quality of life and functional independence. Further more the economic burden of peripheral arterial disease is substantial.

HISTORICAL REVIEW

HISTORICAL REVIEW

- Ulcers of the upper extremity is an old disease described as early as 1500 BC in the ancient Egyptian papyrus.
- Several causes for ulceration of hand have been put forward even from stone age period ranging from Bad evil, Black bile to Honey like urine in diabetes.
- The term DIABETES meaning “to pass through” was first coined by Aretaeus in second century AD.
- The sweet taste of diabetic urine was noted in fifth-sixth century AD by Indian physician Susrutha and called it as “madhu meha”.
- The term DIABETES MELLITUS was coined by John Rollo in eighteenth century.
- Claude Bernad discovered the metabolism of Diabetes in nineteenth century.
- Insulin was discovered at the University of Toronto in 1921 by Fredrick G Banting, a surgeon and Charles H Best, a student assistant.
- Several descriptions of amputations, dates back to Hippocrates. The technique was popularized by Ambrose pare – 1575.
- Yuoung and Lowdhan in 1679 developed flap method of amputation.

- Thromboangitis obliterans, a common disease in smokers, causing multiple small vessel occlusions was described by Leo Buerger in 1908.
- The patter of circulation was described by William Harvey in 1628.
- Recent advances include: Balloon angioplasty for arterial occlusion, Value repair for venous disorder. Growth factor for rapid wound healing.

AIM OF THE STUDY

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1. To study the epidemiology of non-traumatic ulcer of upper limb.

2. To discuss about the various methods of treatment, their merits and demerits and to arrive at a conclusion regarding the method of treatment most suited for our conditions.

3. To study the comments organism causing infection and sensitive drug in patients with diabetic ulcer of upper limb.

METHODS AND MATERIALS

METHODS AND MATERIALS

The clinical material for the study consisted of 50 cases of Non traumatic upper limbs ulcer patients admitted in surgery wards of Govt. Rajaji Hospital during 2006-2008.

The patients were selected with following criteria.

1. only Non traumatic ulcer patients are selected.
2. Thermal burns, scalds are excluded
3. Superficial infections of skin are excluded.
4. Patients with Hansen's disease leading to trophic ulcer are excluded.

Of the 50 patients included in the study, the relevant data were collected as follows and recorded.

In the proforma, age of patient, sex, occupation, complaints and history in details were obtained with information about DM, HT, TB, IHD in past. Personal history about smoking, STD, occupation were enquired. Family history about connective tissue disorder were enquired.

Patients were examined in details about the general condition like anaemia, Jaundice, Xanthelesma and BP and Peripheral pulses.

The affected part with the ulcer was examined in detail for all the features of an ulcer.

THE FOLLOWING INVESTIGATIONS WERE CARRIED OUT AS PER THE NEED OF THE STUDY

Urine : Albumin, Sugar,

Blood : Haemoglobin, total count, differential count, peripheral smear and ESR.

Blood : sugar, urea, VDRL

Serum : creatinine, cholesterol

Pul c/s: in diabetic ulcer

Colour dropper study : in patients with vascular ulcer.

X-ray neck: To demonstrate bony abnormalities.

X-ray of the affected part : To look for osteomyelitis of underlying bone.

Edge biopsy : in suspected malignant ulcer.

The mode of treatment given whether conservative or surgical, complications and follow up were recorded in the proforma.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Chronic Ulcers of upper extremity represent a clinical challenge. Successful treatment requires a thorough understanding of the pathologic process, surgical debridement of devitalized tissue and updating various modalities of treatment. Failure to expeditiously recognize the cause, pathology and associated infectious process may lead to devastating consequences, including limb sepsis, amputation and death.

Ulcer: is a break in the continuity of the covering epithelium – skin or mucous membrane.

Pregenegrane : is the changes in a tissue which indicate that its blood is so precarious that it will soon be inadequate to keep the tissue alive.

Gangrene : is the death of macroscopic tissue with (or) without superadded putrefaction.

Dry gangrene : occurs when the tissue are desiccated by gradual slowing of blood stream.

ARTERIAL ULCER – ZONE OF DEMARCATION

Wet gangrene : occurs when venous as well as arterial obstruction present and is characterized by infection and putrefaction.

Zone of demarcation: between the truly viable and dead or dying tissue is indicated on the surface by a band of hyperemia and hyperasthesia.

Classifications of ulcers of upper limb.

I. Clinical classification:

a). Spreading ulcer:

Surrounding skin of the ulcer is inflamed and the floor in covered with profuse and offensive slough without evidence of granulation tissue.

b). Healing ulcer:

Floor of the ulcer is covered with healthy granulation tissue has sloping edge, reddish with granulation and margin is bluish with serous discharge.

c. Callous (or) chronic ulcer

Ulcer shows no tendency towards healing, has pale granulation tissue and indurated.

ARTERIAL ULCER – DRY GANGRENE

II. Pathological classification:

a) Non specific Ulcers:

i). Traumatic ulcer:

Mechanical eg: Dental ulcer from jagged tooth.

Physical : eg: Electrical or x-ray burns.

Chemical : eg: caustics.

ii). Arterial Ulcer

iii). Venous ulcer

iv). Neurogeic ulcer (Trophic Ulcer)

v). Cryopathic ulcer

Due to systemic disease - Anaemia, Diabetes, Rheumatoid arthritis, gout, martorell's ulcer, Bazin's ulcer.

b). Specific ulcer:

Tuberculosis, syphilitic, actinomycosis, tropical ulcer, meleney's ulcer.

C. Malignant ulcer:

Epithelioma, Rodent ulcer, malignant melanoma.

HEALING DIABETIC ULCER

Aetology of upper extremity ulcers:

I. Atherosclerosis

a). Atherosclerosis obliterans

b). Embolization

1. Cardiac

2. Atheromatous emboli.

II. Arteritis

A). Collagen disorder.

1. Scleroderma

2. Rheumatoid arteritis

3. Systemic lupus erythromatosus

4. Poly arteritis

B). Allergic necrotizing arteritis

C). Takayasu disease

D). Buergers syndrome

E). Giant cell arteritis

III. Blood discrasias

A. Cold agglutinins

B. Cryoglobulins

C. Poly cythemia

IV. Drug induced occlusion

1. Ergot poisioning
2. Drug abuse
3. Dopamine induced ischemia
4. chemotherapeutic agents

V. Occupational trauma

A. Vibration syndrome

B. Hypothenar hummer syndrome

C. Electrical burns

D. Pirching a baseball

E). Playing a musical instrument.

VI. Thoracic outlet syndrome

VII. Congenital arterial wall defects

VIII. Trauma

A). Iatrogenic cathetor injury

1). Cardiac catheterization

2). Arterial blood gas and pressure monitering

3). Arteriography

IX. Renal transplantation and related problems.

A. Azotemic arteriopatty

B. Haemodialysis shunts

X. Aneurysms of the upper extremity

Arteries of upper limb:

Rt subclavian artery begins from brachio cephalic trunk (innominate artery) where as left subclavian artery arises directly from the arch of Aorta. From underneath the sterno clavicular joint artery arches over the plura and apex-of lung about 2.5cm above the clavicle and then reaches the Lateral border of first rib to continue as axillary artery.

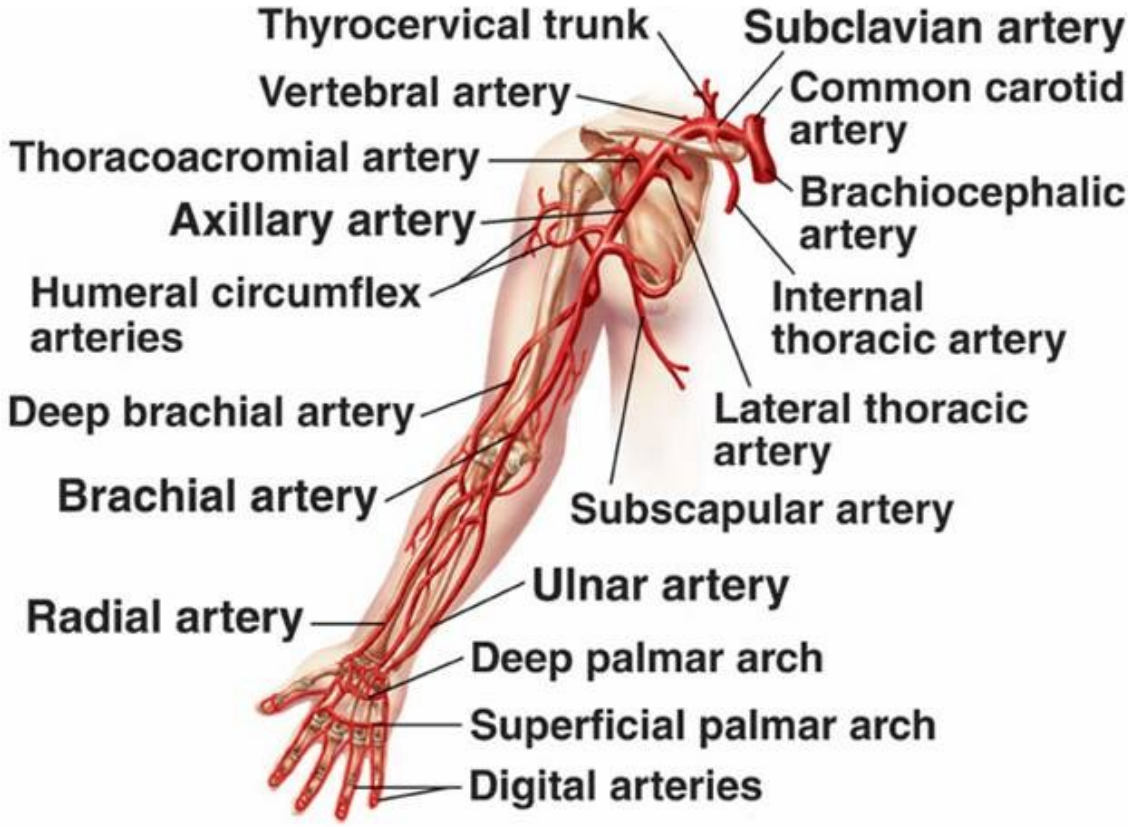
Subclavion artery is divided into three parts by scalenus anterior muscle.

Axillary artery is divided into three parts by pectoralis minor muscle.

As the lateral border of teres major muscle it enters the arm and continues as brachial artery.

About 2.5m below the crease of the elbow joint, it bifurcates into radial and ulnar arteries which run is the forearm.

ARTERIES OF UPPER LIMB



Ulnar artery forms the superficial Palmar arch which is completed by superficial Palmar branch of radial artery.

Radial artery after passing through the anatomical snuff box enters the dorsum of hand and first intermeta carpal space to form deep Palmar arch. It is completed by deep Palmar branch of ulnar artery and is 1cm proximal to superficial Palmar arch.

Arterial Ulcer :

Arterial ulcer are due to ischemia of the skin from peripheral arterial disease and poor peripheral circulation Atherosclerosis of the peripheral arteries is the commonest cause of the condition. It is due to trauma (or) infection that destroys the skin, which fails to heal because of poor arterial supply.

The ulcer is generally accompanied by claudication & ischemic pain. These ulcers are punched out with distraction of the deep fascia. The tendons, bone or underlying joints may be exposed in the floor. Peripheral arterial pulses are feeble or absent. Presence of ischemic charges can be detected eg. Pallor, dry skin, loss of hair, fissuring of nails etc.

ARTERIAL ULCER WITH GANGRENE IN FINGER TIPS

Buerger's disease (Thromboangitis obliterans)

It is a inflammatory reaction in the arterial wall with the involvement of the neighbouring vein and nerve, terminating in thrombosis of artery. It is more common in smokers and lower socio – economic groups.

The diseased artery is usually surrounded by a dense fibrotic reaction which incorporates the adjacent vein and less often the neighboring nerve. The lesions in Buerger's disease are segmental and usually begin in arteries of small & medium size. Both upper & lower extremities are affected. TAO has also been described in the G.I tract, lungs, heart and male genitalia.

The acute lesion consists of acute arteritis and periarteritis, acute phlebitis and periphlebitis. The wall of the vessels are invaded by polymorphonuclear leucocytes. Thrombosis occurs within the vessels with occlusion of the lumen. Giant cells are often present in the thrombus. Small micro abcesses buries within the thrombus may present. These abcesses have a central focus of polymorpho nuclear leucocytes. Only a segment of the vessel is involved, which may be long or short.

In chronic lesion, the artery and the vein are bound together by fibrous adhesions. The Nerve may also be involved in the same adhesion. Involvement of the nerve is responsible for agonising pain. The thrombus

TAO AFFECTING BOTH UPPER LIMBS

shows fibroblastic activity and endothelial proliferation. The thrombus is now organized into fibrous tissue. The internal & external elastic laminas of the artery are frequently much thickened.

There may not be any specific finding till gangrene develops. The peculiar feature is that the ischemic area is usually sharply demarcated with relatively good circulation in adjacent tissue. Before gangrene there may be signs of chronic tissue ischemia. These include lose of hair from the digits, atrophy of the skin and brittle nails. Gradually there may be ulceration or gangrene of the digits, commencing in the distal portion of the digit near the nail and gradually extend proximally to involve whole of the hand.

Raynaud's disease

The arterioles penetrate the dermis at right angle, with an irregular reticulate pattern & end in a capillary network. In Raynaud's disease, vasospasm occurs with such severity that dermal circulation momentarily ceases with the production of severe pallor. If the vasospasm is less severe, with showing but not cessation of dermal circulation, cyanosis appears. After some minutes of pallor, the capillaries dilate due to hypoxia and accumulation of metabolic products of regional anaerobic metabolism. This is followed by a slight relaxation of the arteriolar spasm with entry of small amount of blood into the dilated capillaries. This is rapidly deoxidised and this gives rise to cyanosis. This results from sluggish flow of blood with

an increase in the percentage of reduced Hemoglobin in the capillaries. When the vasospasm subsides, a reactive hyperreamia with vasodilatation develops due to accumulation of tissue metabolite during the anoxic period and this reduces redness or rubor.

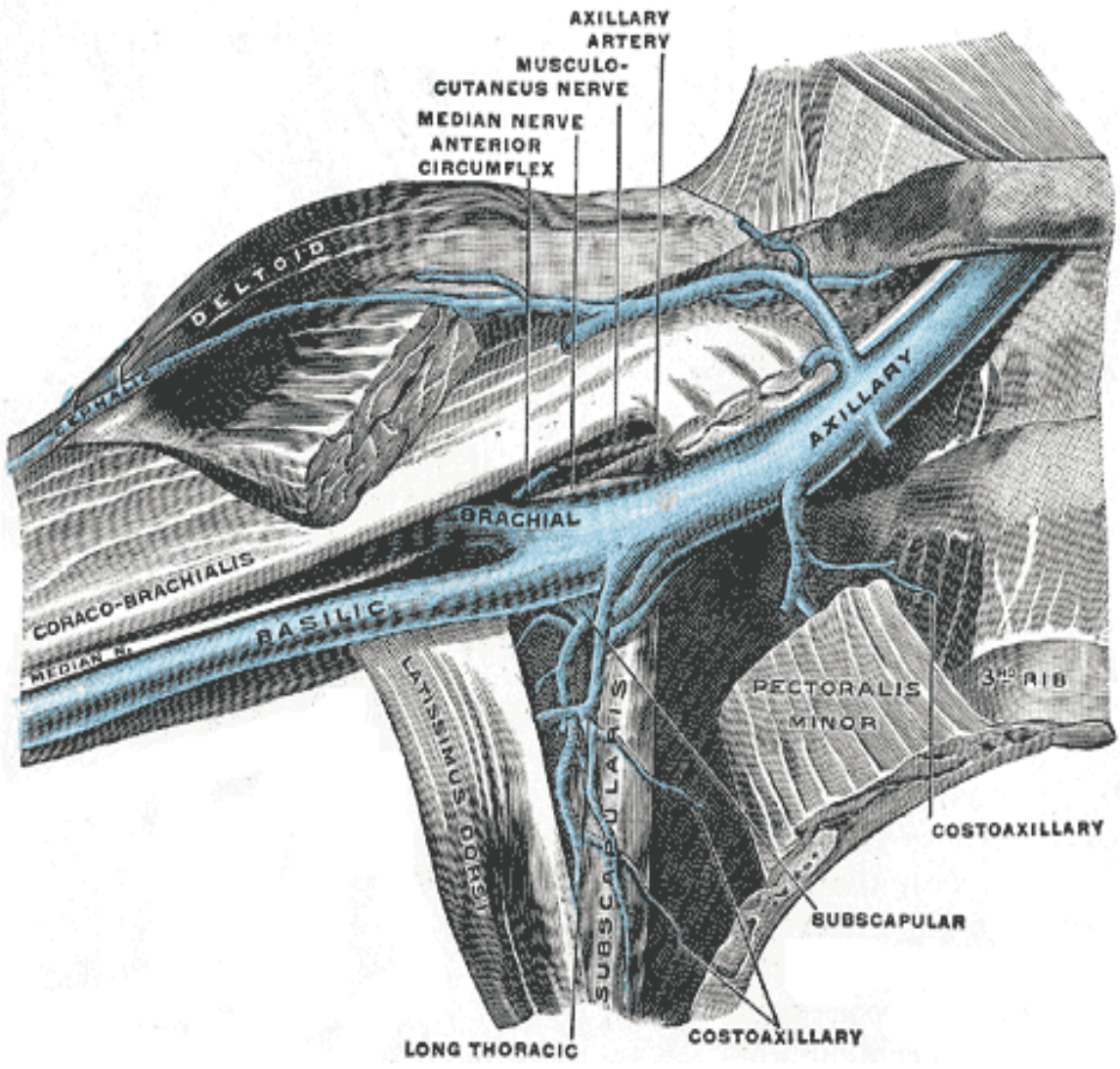
Use of vibrating tools or exposure to chronic cold play a role in this disease. There are many theories for the disease.

- It may be due to increased sensitivity of the arterioles or hyperactivity of the sympathetic nervous system.
- There is decreased hand and finger blood flow at room temperature and a striking additional decrease with cooling below the level of 18C at which point the digital artery completely closes.
- It may be due to abnormal adrenergic receptors that become increasing sensitive to stimulation following cold expose.
- Raynaud's disease is common in younger women & is commonly bilateral. Three stages are distinctly observed with expose to cold (or) emotional disturbances.
- Stage of local syncope : with expose to cold the digital arterioles go into spasm and the decreased blood flow is evident by pallor or blanching (stage of blanching). This change starts at the tip of the finger and gradually spreads toward the base.

- Stage of Local Asphyxia: With gradual warming, there is slight relaxation of the arterioles. Small amount of blood passes to the capillaries which become dilated due to accumulation of anaerobic metabolites from the previous stage. Slowly flowing blood becomes easily deoxygenated & the part becomes dusky or cyanosed (stage of dusky anoxia). The fingers remain cold & numb.
- Stage of recovery: As the attack passes off & with more warming, the spasm absolutely disappears & the arterioles relax. The oxygenated blood returns into the dilated capillaries and the fingers become red (stage of red engorgement) & swollen. There may be burning sensation or pain produced by the increased tissue tension within the digits.

In long standing cases, the fingers gradually waste, especially the pulps, which become thin & pointed. Small scars may appear following necrosis of small areas of skin. Small and painful ischemic ulcers may be seen on the finger tips. Repeated infections (Paronychia) are common around the nails. There are painful & slow to heal.

ANATOMY OF THORACIC OUT LET



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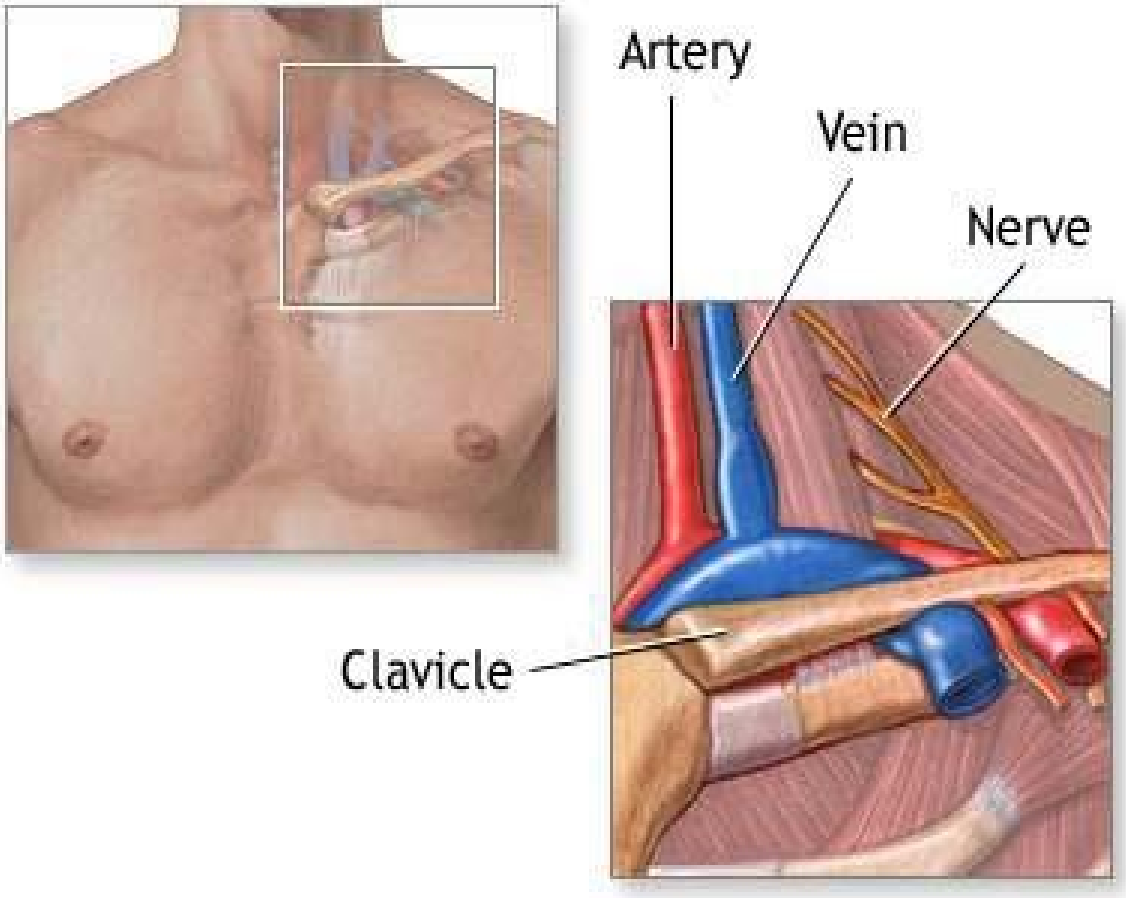


Surgical anatomy of the thoracic Outlet:

The superior thoracic outlet is bounded by the manubrium sternum anteriorly, the spine posteriorly and the first rib laterally. At the superior aperture of the thorax, the subclavian vessels and the brachial plexus traverse the cervico axillary canal to reach the upper extremity.

The cervicoaxillary cancal is divided by the first rib into two parts. Proximal one, composed of costoclavicular space and the distal one composed of axilla.

The proximal division is more critical for Neuro vascular compression. It is bounded superiorly by the clavicle and inferiorly by the first rib, anteromedially by the costoclavicular ligament and posterolaterally by the scalenes medius muscle along with long thoracic nerve.

The scalene anticus muscle divides the costo clavicular space into two compartments. The anterior one containing subclavian vein and the posterior one containing the subclavian artery and brachial plexus. This compartment is bounded by scalene anticus anteriorly, scalene medius posteriorly and the first rib inferiorly is called scalene triangle.

Thoracic outlet syndrome:

The syndrome is caused by compression of the brachial plexus or subclavian artery and/or vein in the region near the thoracic outlet. The symptoms may arise from neural, vascular or combined compression.

Presence of cervical rib or fractures of clavicle or first rib with bony calus or a wide scalene anticus muscle may narrow the space in the interscalene triangle.

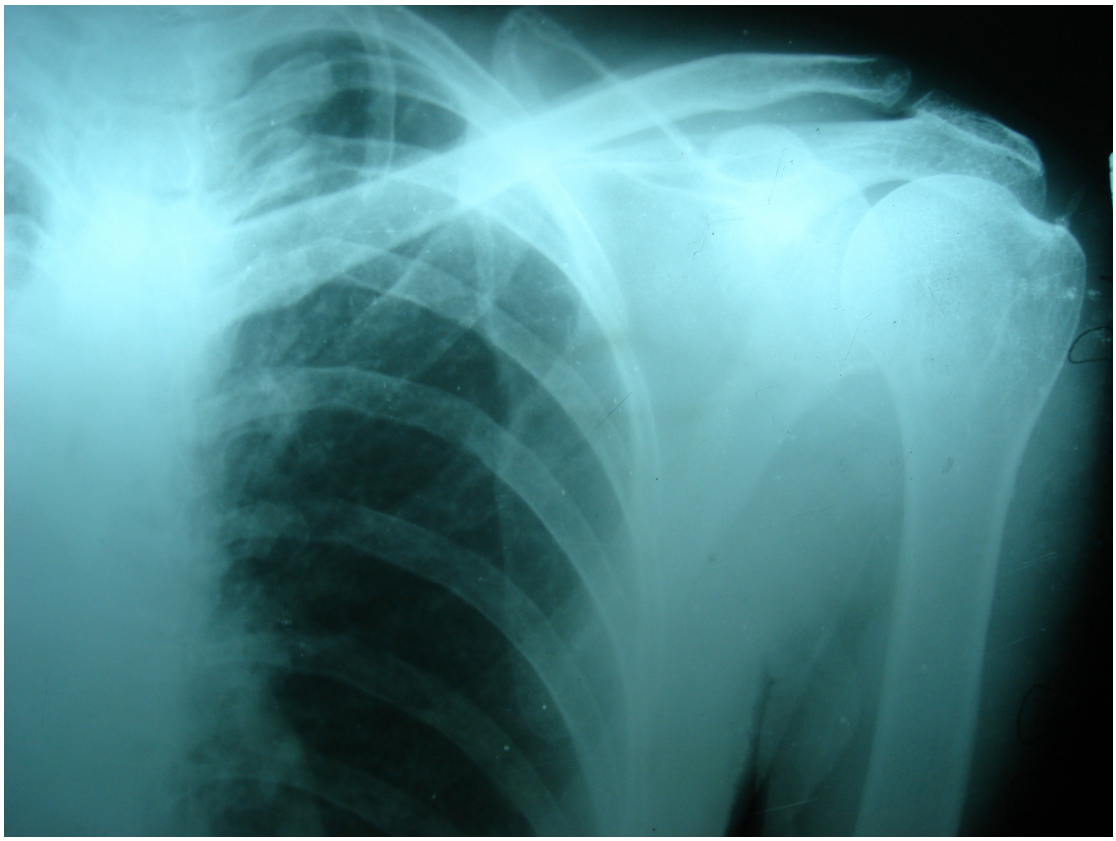
Vascular symptoms may be intermittent from compression or temporary occlusion of the subclavion artery or due to intermittent vaso constriction, similar to that seen in Raynaud's phenomenon.

Arterial compression may produce pain, numbness, paraesthesia, coldness & weaknes of the arm or hand. Distal embolisation may cause Raynaud's syndrome, which in late cases may produce digital ulceration & even gangrene.

Trophic ulcer (Neurogenic ulcer):

These ulcers develop as a result of repeated trauma to insensitive part of the body. Neurological disturbances in the form of sensory loss is the cause behind the ulcer formation. The ulcer has punched out edge with

X-RAY SHOWS CERVICAL RIB



slough in the floor with tendon and even bone being exposed. The base is indurated surrounding skin has no sensation. The causes may be spinal cord injury, Hansen's disease, peripheral neuropathy and diabetes.

Cryopathic ulcer:

It results from the exposure to intense cold chilblains results from exposure to chill weather that results in blister formation and ulceration. Frost bite occurs when any part of body is exposed to wet cold below freezing point, which causes arteriolar spasm, freezing of tissues and denaturation leading to gangrene and full thickness loss of skin.

Diabetic ulcer:

Three factors play predominant role in the genesis of diabetic ulcer.

1. Diabetic neuropathy
2. Diabetic atherosclerosis resulting in ischemia.
3. Hyperglycemia.

The neuropathic factor impairs sensation and then favors the neglect of minor injuries and infections. So inflammation and damage to the tissues are ignored.

DIABETIC ULCER

Theories for diabetic neuropathy:

1. Metabolic theory:

The increased blood Glucose level leads to increased formation of sorbitol, which in found to be toxic to nerves. Reduction in the phorphoinositol leads to reduced myelin production.

2. Vascular theory:

This theory suggests the neuropathy is due to microangiopathy leading to reduced blood flow to the nerve sheath bundles leading to chronic ischemia of progressive nerve damage.

Diabetes is associated with microangiopathy as well as macroangiopathy since it hastens atheroselerosis.

Excess of sugar in the tissues favours growth of organism and lowers resistance to infection. Osteomyelitic changes can occur in the bone due to spreading infection of bone necrosis.

The ulcer are characterized by rapid progression and they spread along all the tissue planes damaging fascia, tendons, muscle, and bones often with mild local and general symptoms.

Grading of diabetic ulcers are:

Grade O : Pre ulcerative lesion

I : Superficial ulcer

II : Deep ulcer with no tendon or bone involvement.

III : Deep ulcer extending to tendon or bone

IV : Deep ulcer with gangrene

V : Infected diabetic ulcer.

Anaemia:

Congenital Haemolytic anaemia like spherocytosis, sickle cell anaemia predispose to ulcer formation. Sickle all anaemia predisposes to ulcer formation due to occlusion at micro vascular levels. Stasis and hypoxia predispose to occlusion of vessels by sickling leading to infarction and ulcer formation. It is associated with Jaundice and gall stone disease due to chronic haemolysis with positive family history. The ulcer is surrounded by block pigmentation.

Ulcers due to specific infections:

1). Tropical ulcer

2). Tuberculosis ulcer

3). Syphilitic ulcer

4). Meleney's ulcers

5). Yaw's

Tropical ulcer:

It is a rare ulcer, which occurs following trauma or insect bite. It is caused by Vincent's organism (Fusobacterium and borrellia). It commences as a papulopustule which bursts in a couple of days to form a painful, foul smelling ulcer. The edge of the ulcer in typically undermined due to considerable infiltration in the surrounding skin. The spreading process ceases after a few weeks, resulting in a non healing ulcer which refuses to heal for even months. In few cases, it assumes phagedemic (eating) characteristic resulting in wide spread destruction of the soft tissue necessitating even an amputation. Rarely squamous carcinoma may supervene. After a long period of healing it leaves behind a paper like pigmented scar.

Tuberculous ulcer:

This is commonly results from bursting of caseous lymph node, but can also develop when cold abcess from bone and joint tuberculosis breaks out on the surface. The ulcer is irregular in shape and has a undermined thin edge. The floor is covered with pale granulation tissue with apple jelly appearance, with thin serosanguinous discharge. The base is not indurated. The surrounding skin shows bluish black pigmentation. The regional nodes are enlarged, non-tender, firm and matted. There may be systemic evidence of tuberculosis of lung or other part of the body.

Lupus vulgaris

It is a form of cutaneous tuberculosis occurring in young adults which starts as cutaneous nodule which gradually turns into ulcer. The ulcer remains active in periphery and spreads outward's. Whereas in the center, they gradually heal. Due to its destructive nature, it is called 'Lupus' which means 'wolf'. Squamous cell carcinoma may grow from the scar of lupus vulgaris to form Marjolins ulcer.

Meleney's ulcer:

It is due to symbiotic infection of micro-aerophilic non hemolytic streptococci and staphylococci.

It occurs de novo in association with ulcerative colitis or as a complication of a previously existing ulcer. It has undermined edges and foul smelling abundant granulation tissue with copious seropurulent discharge. It is surrounded by deep purple zone, which in turn is surrounded by outer zone of erythema. These ulcers are very painful, tender, show tendency to spread and make the general condition of the patient to deteriorate if not treated.

Malignant ulcers:

Squamous cell carcinoma (Epidermoid carcinoma / epithelioma)

It arises from the prickle cell layer of the skin. It is mostly seen after 40 yrs years of age. It begins as a small nodule, which enlarges and gradually the center becomes necrotic and sloughs out and thus ulcer develops. Such an ulcer is oval or circular in shape, but size varies considerably. The edge of the ulcer is raised and everted, floor is covered by necrotic tumor tissue, serum and blood. Base of the ulcer is indurated which is the pathognomonic sign of epithelioma. In later cases, it becomes fixed due to involvement of the deeper structures. Regional lymph nodes are almost always enlarged either due to metastasis or secondary infection. Metastatic mode is hard and fixed.

Basal cell carcinoma (rodent ulcer)

It originates from the basal layer of the rete Malpighi of the skin. It is a low grade malignancy most commonly seen in white skinned people, often involving, the sun exposed area namely the face.

It start as a small brownish red nodule with translucent colour and shiny surface showing a network of capillaries. At this stage it is diagnosed due to its hardness, painleseness and presence of capillaries, later the tumors ulcerates with a well defined hard and raised edge with beaded appearance.

It

SQUAMOUS CELL CARCINOMA

infiltrates into the surrounding as well as deeper tissues, even upto the bone and hence the name rodent. At first it may itch, but at a later stage it may be painful if it has involved any nerve.

Dissemination by lymphatics or blood vessels does not occur, so that regional nodes are not enlarged.

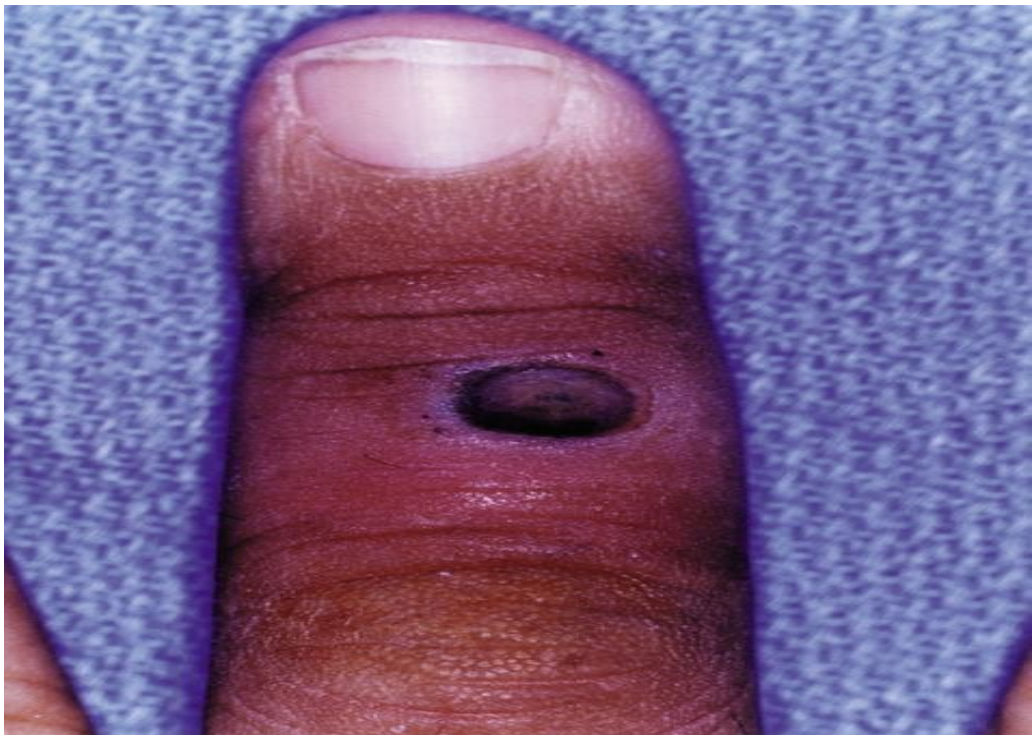
Malignant melanoma:

It is a malignant tumour of melanocytes which originate from the neural rest and so ectodermal in origin. It may occur de novo or most often from benign mole. The following points suggest malignant conversion of a mole.

- Asymmetry
- Border irregularity
- Colour variation
- Diameter more than 6mm size
- Elevation.

The predisposing factor is exposure to sun light, functional and dysplastic nevi and immuno suppression. It is more common in females and occurs between 30-50 years.

MALIGNANT MELANOMA



The size and appearance vary clinically because it has vertical and radical growth. The depth of invasion can be measured by clark's level of invasion or by Breslow's index. It can have satellite nodules and in transit metastasis due to lymphatic spread. Regional nodal metastasis and visceral metastasis to liver, lung, skin, gut and bone are common.

Marjolin's ulcer:

This is a squamous cell carcinoma arising from a long standing benign ulcer or scar. It is slow growing and is less malignant. Its edges are not raised or everted. It is absolutely painless and lymphatic metastasis is unusual as the nerves and lymphatics are already destroyed or occluded by previous chronic lesion of skin. Unlike squamous cell carcinoma it is radio resistant.

Vasculitis:

Heterogenous group of disorders characterized by inflammation and damage of blood vessels followed by thrombosis and ischemic manifestations in the tissue supplied by the blood vessels.

Hypersensitivity vasculitis : (Leucocytoclastic vasculitis):

It may be caused by connective tissue diseases (systemic lupus erythromatosus, Rheumatoid arthritis), Hepatitis – B infection, Drugs

GANGRENE DUE TO VASCULITIS

(Penicillin's, phenothiazines, phenyl butazones, Propylthiouracil) and Hematologic diseases (Cryoglobulinemia, Paraprotenemia, multiple myeloma), capillaries and venules are commonly affected.

Polyarteritis nodosa:

Exact cause is unknown. 20% of cases associated with hepatitis B infection. Medium size and small arteries are affected. On histo pathological examination, characteristic areas of fibrinoid Necrosis and thrombosis seen.

Takayasu's arteritis:

Is a Nonspecific inflammatory process of unknown etiology affecting segmentally the Aorta and its main branches. The main pathologic process is panarteritis probably first affecting the adventitia of vaso vasorum. Later the media is affected with a loss of elastic fibres. The Aetiology remains obscure. But the current consensus favours hypersensitivity or autoimmune reaction.

Ergot Poisoning:

Ergot compounds produce intense vasoconstriction because of direct stimulation of the alfa receptors in the vessel wall. In prolonged ischemia due to ergot poisoning, thrombosis with tissue necrosis may occur. This is often bilateral and tends to be symmetric. Occasionally focal spasm in seen.

Radiation injury:

Irradiation may cause damage not only to small vessels but also to large arteries such as the subclavian. These changes include endothelial proliferation, degeneration of cells of the media with subsequent cystic medial necrosis and fibrosis.

There are 3 stages following irradiation. The first is at 5 yrs following radiation, when most patients present with mural thrombosis. The second stage occurs 10yrs after irradiation, when patients present with symptoms caused by fibrobic occlusion of the irradiated vessel. The third stage is very much later (20-25yrs or more following irradiation) when the lesion involves periarterial fibrosis together with accelerated artherosclerosis.

Collagen disease:

Collagen disease includes scleroderma, Rheumatoid arthritis, systemic Lupus erythromatosis and dermatomyositis. It is characterized by generalized connective tissue damage with an increase in the amount of collagen in skin, muscle, tendons, fascia and viscera. When the characteristic histologic changes of fibrinoid degeneration and intimal thickening occurs in the blood vessels, signs of ischemia can occur.

Blood dyscrasias:

Cold agglutinins, cryoglobulin, and polycythemia vera are the most common form of blood dyscrasias of the arteries of the hand. The cause of small artery occlusion is generally thought to be local thrombosis or embolism.

Drug abuse:

Vascular complications due to drug abuse may be related to local damage to the artery by unsterile needles leading to infection, false aneurysm formation or arteriovenous firtula. The inadvertent injection of hypertonic solution or powder into the arterial system often causes multiple digital arterial occlusions.

Catheter injury:

With increasing use of diagnostic and therapeutic procedures involving catheterization, damage to radial or brachial artery has become more common, especially when an incomplete palmar arch is not recognized prior to placement of catheter in the radial artery. It causes thrombosis, embolization, or dissection and may cause exercise pain in fore arm or cold sensitivity of the involved extremity.

Hypothenar Hammer syndrome:

The predisposing factor in the development of hypothenar hammer syndrome is the repetitive use of the palm is activities that involve pushing, pounding or twisting. The anatomic location of the ulnar artery in the area of the hypothenar eminence places it in a vulnerable position. When the area is repeatedly traumatized, ulcer artery occlusion or aneurysm formation can occur.

Azotemic arteriopathy:

In CRF, calcification may affect the digital arteries of the hand causing gangrene of the digits. It is characterized by calcification of the media of the digital arteries.

Management of upper limb ulcers:

Investigations:

For proper management of upper limb ulcers the most important thing is thorough clinical examination and few special investigation for confirmation which are streamlined according to the history and clinical findings.

FINGER DISARTICULATION–WOUND INFECTION

Urine Analysis: If diabetes suspected

Full blood examination:

Blood sugar & renal parameters:

The patient must be screened for diabetes mellitus with urine sugar, random blood sugar and glucose tolerance test if necessary. Renal parameters should be evaluated since renal failure can co-exist with diabetes and renal failure by itself may delay ulcer healing.

Culture and smear of ulcer discharge:

The bacteriology of infective organism causing infection has to be identified for its effective control & fruitful outcome of the treatment. The type of organism causing infection depends on the cause of the ulcer. In diabetic foot, the common organisms are staphylococcus areus, enterococci, gram negative bacilli like E.coli, proteus, klebsiella, pseudomonas and some anaerobes like peptococcus, Bacteriods, clostridium etc. In ulcer due to vascular disorders, infections are often caused by staphylococcus and streptococcus.

Edge biopsy of ulcer:

Edge biopsy is taken from the ulcer if malignancy or infection due to specific organism eg. Tuberculosis is suspected. In malignant ulcer biopsy shows typical epithelial pearls pattern in squamous cell carcinoma,

RAY AMPUTATION – WOUND INFECTION

pallisading manner of arrangement of cells in basal cell carcinoma and nests of melanin containing cells with invasion into dermis and deeper tissues with a typical morphological appearance and increased mitotic figures in malignant melanoma.

Digital subtraction Angiography:

More advanced method of arteriography in which computer subtract the pixels of a first image of the series from subsequent images removing extrenous background and so provides great clarity.

Transcutaneous oximetry:

Transcutaneous measurement of O_2 tension for assessing tissue perfusion has a role in assessment of critical ischemia particularly in diabetic patients with extensive vascular calcification.

Nerve conduction velocity studies (NCU):

For Neuropathic ulcer, the conduction velocity of a nerve is calculated after application of electric stimuli, by measuring the time taken by the action potential created by the stimuli to travel along the nerve. Nerve conduction velocity is useful in detecting sensory & mixed nerve involvement.

Digital Plethysmography:

Can be useful is differentiating proximal from distal arterial obstructive phenomena and also to differentiate primary Raynaud's disease from Raynaud's phenomenon associated with collage vascular disease.

X-ray neck and X-ray chest:

To demonstrate bony abnormalities eg. cervical ribs, bifid first rib, fusion of the first and second rib and clavicular abnormalities and also to show narrowing of the intervertebral foramina by exostosis or tumours.

X-ray hand:

Collagen vascular disease documented by distal phalangeal tuft reabsorption and evidence of soft tissue atrophy with particular loss of pulp dimensions on the palmar surface of the distal phalanges.

Serological rests:

When connective tissue disease is suspected, the following tests may be helpful to arrive at a diagnosis.

a. serum protein electrophoresis
b. Cold agglutinins
c. Rheumatoid factor
d. VDRL
e. Hep 2
f. Antinuclear antibodies
g. Antinative DNA antibodies
h. Total Haemolytic complement.
i. Complement (C_3. C_4)
j. Immunoglobulin electrophoresis
k. Cryoglobulin
l. Direct combs test
m. Hepatitis B scheming

Dopper and duplex USG

CT Neck

MRA (Magnetic resonance angiography)

After confirmation of the cause of the ulcer, the treatment is carried out.

Treatment:

Current methodology in ulcer management is as follows.

Risk factor modifications:

1. Lipid management : Goal is

Serum LDL : < 100mg/dl

HDL : > 35mg/ dl

TG : <200 mg/dl

2. Weight reduction:

- Physical activity
- Dietory modifications.

3. Smoking:

- Complete cessatioon
- Behaviour modification
- Counselling
- Nicotine analogues.

4). Blood pressure :

- Weight control
- Physical activity
- Sodium restriction
- Antihypertensive drugs.

5). Physical activity:

a). Walking

b). Cycling

c). Jogging

d). Life style modification.

Dressings:

a). Alginates

b). Hydrocolloids

c) Hydro gels

d). films

e). foams

f). Medicated dressings

Physical therapies:

a). Hyperbaric Oxygen

b). Lasers

c). Magnetic stimulation

d). ultrasound Stimulation

e). Vacuum devices

f). Warming

Biological therapies:

a). Cytokines

b). growth factors

c). Protease inhibitors

d). Cadaver Skin

e). Epidermal allograft

f). Dermal allograft

Pharmacological therapies:

1). Aspirin:

Inhibits prostaglandin synthesis which prevents formation of platelet aggregating thromboxane A_2.

2). Clopidogrel:

Selectively inhibits ADP binding to platelet receptor & subsequent ADP mediated action of glycoprotein GpIIb/IIIa complex there by inhibiting platelet aggregation.

3). Pentoxifyline:

Improves blood flow by increasing red blood cell deformability which decreases viscosity of blood.

4). Simvastatin: (Antilipemic agent)

Competitively inhibits HMG-COA which catalyzes the rate limiting step in cholesterol synthesis.

Exercise and physical activity:

Physical activity is known to promote and maintain health, including lower BP, inflammation markers and lipids, reduced prevalence of over weight and depression. Higher level or longer duration of physical activity is related to longer survival and lower risk of death from cardio vascular disease among peripheral arterial disease patients.

Smoking Cessation:

Smoking cessation is a key modifiable risk factor for increasing survival and reducing the potential for disease progression. Smoking increases the risk of amputation, the risk of developing coronary heart disease or stroke and mortality among peripheral arterial disease patients.

Dietary intake:

Olive oil rich in both monounsaturated / poly unsaturated fatty acids, as well as flavonoides and terpena. It is known that poly unsaturated fatty acids favorably influence serum cholesterol concentration and endothelial function, while flavonoids and terpenes function as antioxidants.

AE AMPUTATION – GUILLOTINE METHOD

Vitamin E also contains antioxidants and anti inflammatory properties and inhibits platelet aggregation. Cardio protective nutrition interventions which include reduction in saturated fat, trans fat & dietary cholesterol. The

dietary pattern should be individualized to provide a fat intake of 25-35% of total calories, <7% of total calories from saturated fat and trans fatty acids and <200mg of cholesterol per day.

Surgical Interventions

a) Debridement
b) Drainage
c) Excision
d) Skin grafting
e) Revascularization
f) Reconstruction
g) Amputation

The current concept of wound management is multidisciplinary approach. The general surgeon can give support for wound debridement and delayed suture; split thickness grafts or flap cover may require plastic surgeons help. A vascular surgeon provides help with revascularization, some times with the help of interventional radiology, & to undertake vascular surgery when indicated. All this is supported by endocrinologists

AE AMPUTATION – SHOWING FLAP NECROSIS

(diabetic ulcers), care of the elderly by physicians and nurse specialists. Thus wound management requires professional expertise from a wide range of background.

Arterial Ulcers:

General measures like absolute abstinence from smoking, regular use of analegics, Haemorheological agents like pentoxyphyline, Dextran-40, Antiplatelet agents, varodilators like calcium channel blockers, PGE2, prostacyclin, antidepressants especially tricyclic antidepressants, simple dressing and removal of gangrenous part has to be carried out.

The presence of ulcers means that there is severe degree of ischemia and hence local treatment of the ulcer is unlikely to be effective unless the arterial supply can be improved either by,

Direct arterial surgery

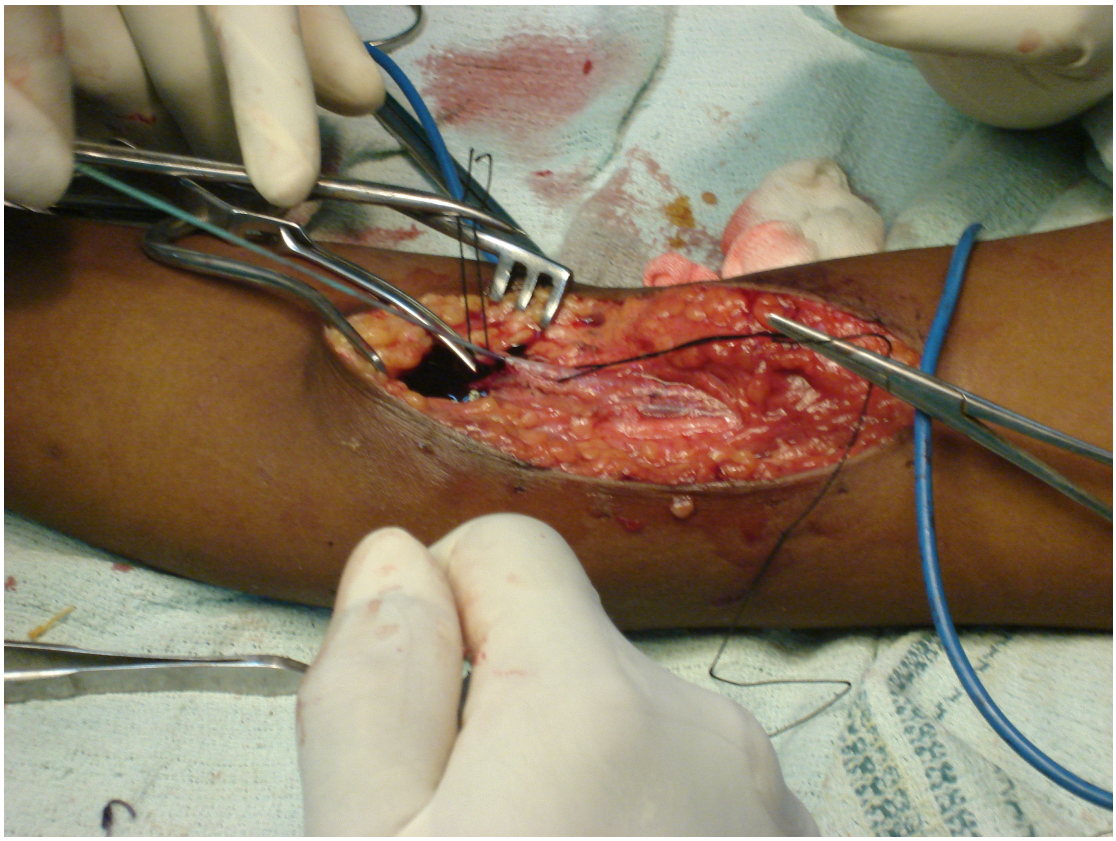
Ballon angioplasty

By pass operation

End arterectory

Direct arterial surgery is attempted in the pathology of large and medium sized vessels. Before arterial surgery is undertaken, arteriography must be done.

EMBOLECTOMY



Trophic ulcer:

a) Treatment of the cause:

The prime cause predisposing to trophic ulcer formation has to be removed Viz – avoiding pressure points and alcoholism, correcting diabetes mellites and vitamin deficiency.

b) Treatment of the ulcer:

Trophic ulcer are best treated by rest and simple dry dressing. Persistent ulcer will require excision of the ulcer with closure of

defect by primary suture, skin graft , local flap cover or distant free flaps.

Cryopathic ulcer:

1) Protection from cold and warming of whole body.
2) Vasodilator drugs may be tried in severe and recurrent cases.
3) Regular dressing and oral antibiotics and analegics
4) Excision of dead tissue and skin grafting
5) Hyperbaric oxygen
6) Amputation

Diabetic Ulcers:

1) **Control of infection:**

Control of infection is the first priority in the management of diabetic ulcers. If there are signs of spreading infection or systems involvement (i.e fever, tachycardia or loss of diabetic control) the patients should be admitted in hospital for intensive treatment with parentral antibiotics. The ususal infecting organisms are stapyloccous aureus, gram negative bacilli and mixed infections.

2) **Control of Blood glucose:**

Control of blood glucose need temporary treatment with plain insulin. With plain insulin, the control of blood glucose is easier and has less complications.

3) **Surgical role:**

The surgical role may involve desloughing of ulcer, drainage of Pus in the tissue planes and minor or even major muputations. The key to success is to remove all dead tissue and to leave the wound open. Amputations are done for removal of dead nectrotic tissues, uncontrolled infection with wet gangrene and uncontrolled osteomyelitis with soft tissue necrosis.

DIABETIC ULCER - SPLIT SKIN GRAFT DONE

Amputations can be performed as an emergency or elective procedure. The level of amputation is dictated by the extent of tissue damage; infection and vascularity. The raw area left behind after the control of infection can be skin grafted or covered with flap depending on the site and size.

Ulcers due to specific infections:

Ulcers due to infective etiology have to be managed according to causative organism with antibiotics similar to that of any other site.

Malignant ulcers:

In management of malignant ulcers, surgery, radio therapy, chemotherapy and other options available here to be considered. The stage of the disease, type of malignancy, grade and site dictates the mode of management.

For the primary site wide excision with 2-5 cm clearance is indicated in squamous cell carcinoma and malignant melanoma. In basal cell carcinoma wide excision with 2cm clearance in the best treatment though radiotherapy, cryotherapy, mohs micro surgery can be adopted.

In case of marjolins ulcer wide excision is the best option.

For regional nodes in case of squamous cell carcinoma and malignant melanoma, block dissection for mobile nodes and irradiation with palliative Chemotherapy for fixed nodes is adopted. For in transit metastasis and recurrent melanoma, isolated limb perfusion therapy can be given.

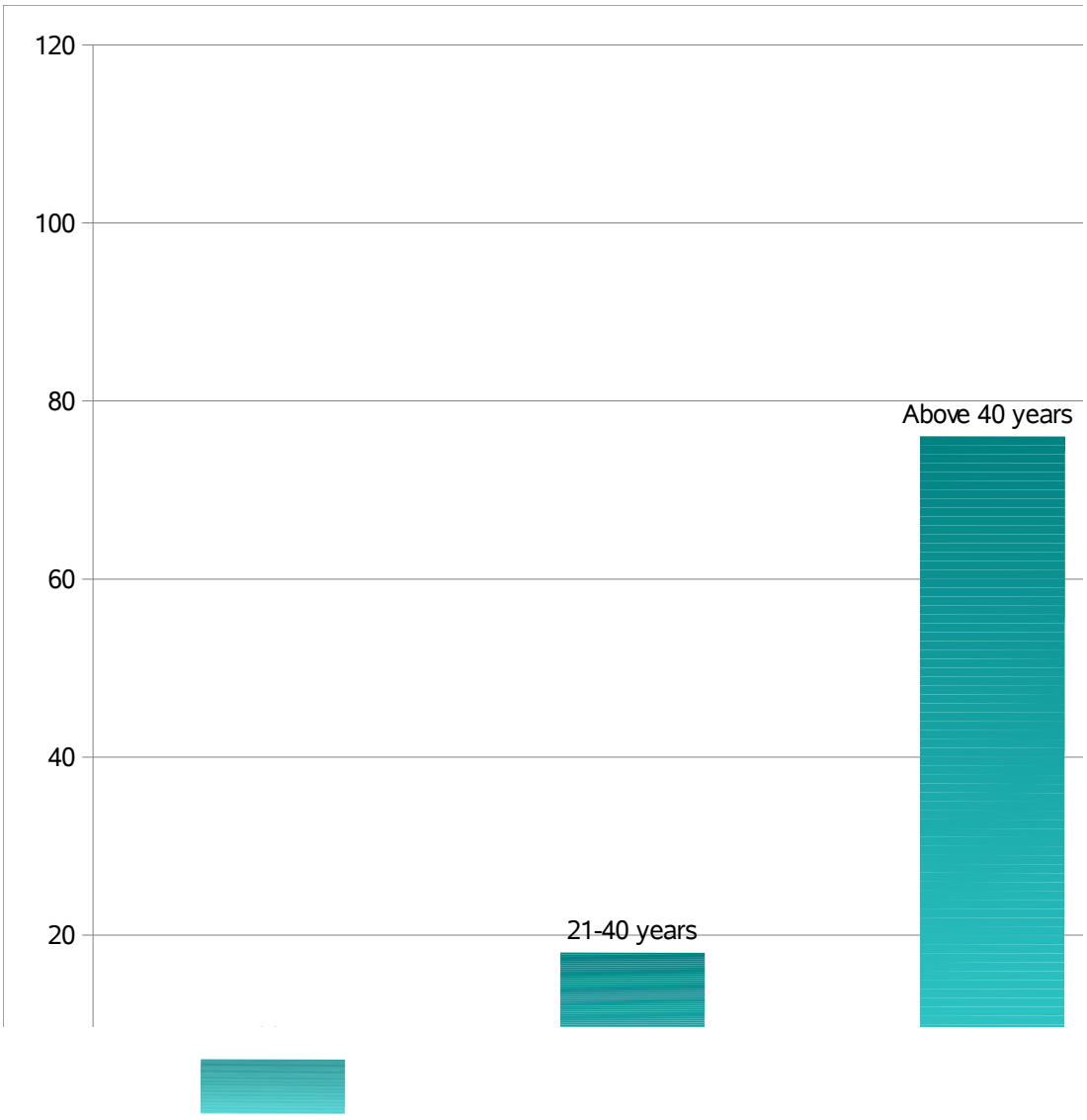
OBSERVATIONS

OBSERVATION

AGE WISE INCIDENCE:

Age Group	No.of patients	Percentage
Upto 20 years	3	6%
21-40 years	9	18%
Above 40 years	38	76%
Total	50	100%

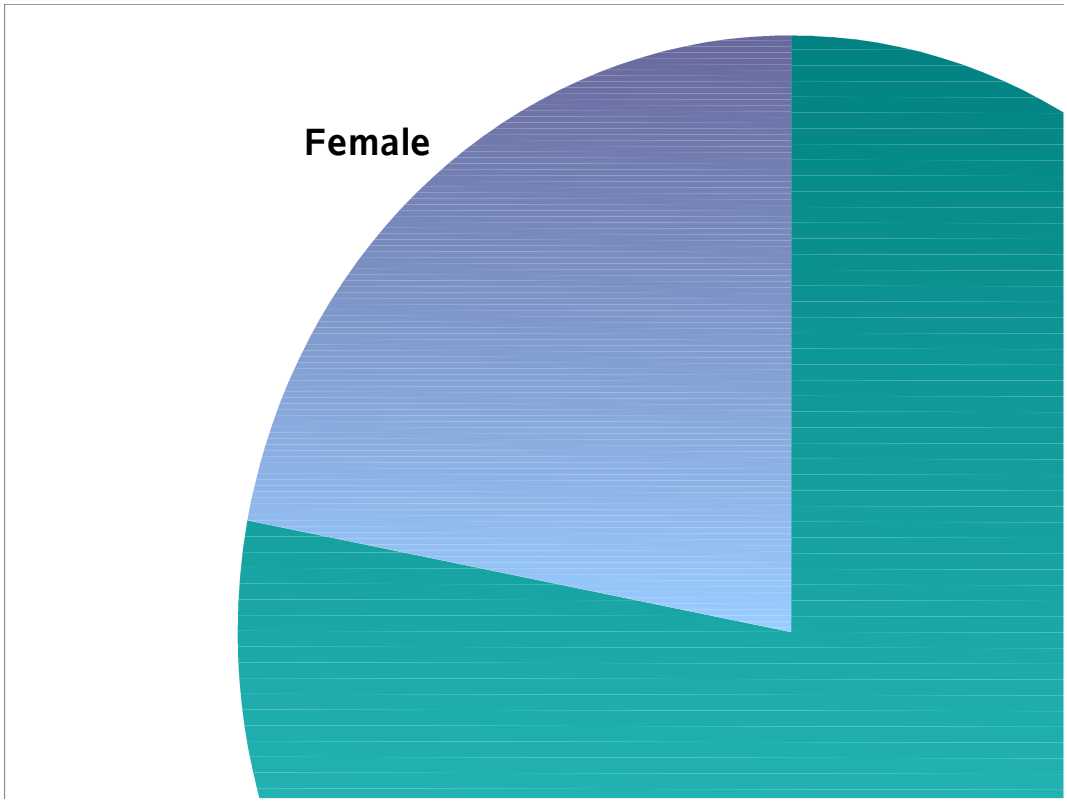
AGE RELATED INCIDENCE



SEX WISE INCIDENCE

Type of ulcer	Male		Female		Total	
	No.	%	No.	%	No.	%
Diabetic	17	34%	5	10%	22	44%
Arterial	14	28%	3	6%	17	34%
Malignant	2	4%	1	2%	3	6%
Trophic	3	6%	1	2%	4	8%
Tuberculous	1	2%	-	-	1	2%
Others	2	4%	1	2%	3	6%
Total	39	78%	11	22%	50	100%

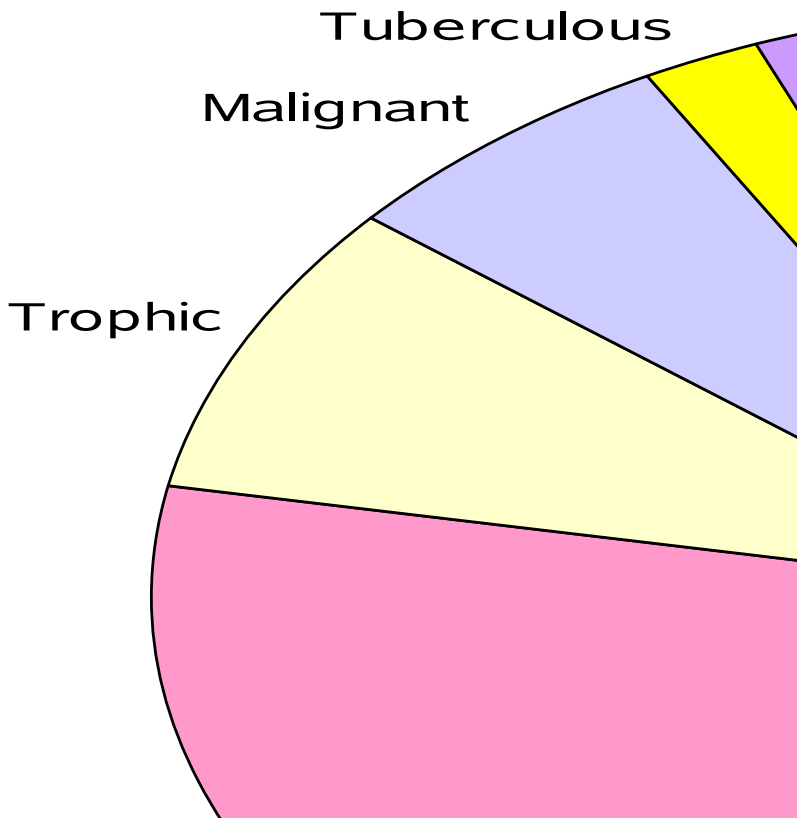
SEX WISE INCIDENCE



INCIDENCE OF VARIOUS UPPER LIMB ULCERS

S. No.	Type of ulcers	No.of cases	Percentage
1	Diabetic ulcer	22	44%
2	Arterial ulcer	17	34%
3	Malignant ulcer	3	6%
4	Trophic ulcer	4	8%
5	Tuberculosis ulcer	1	2%
6	Others	3	6%
	Total	50	100%

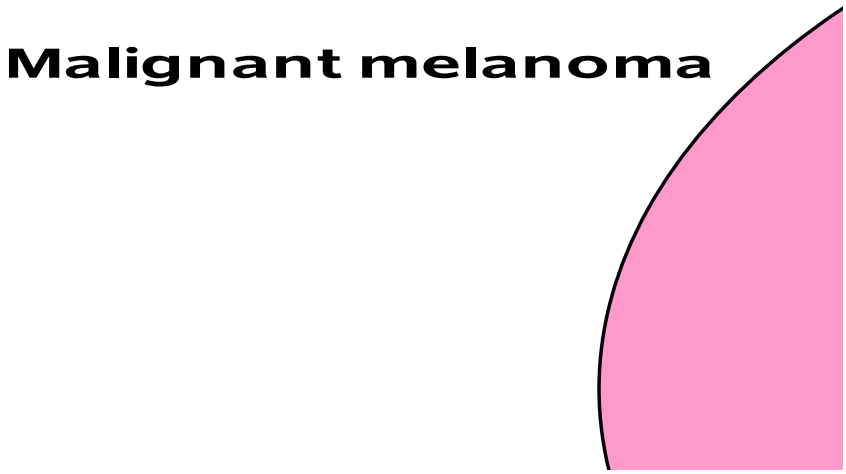
INCIDENCE OF VARIOUS UPPER LIMB ULCERS



BIOPSY REPORT IN MALIGNANT ULCER

Type of malignancy	No.of Patients	Percentage
Squamous cell carcinoma	2	66.67%
Malignant melanoma	1	33.33%
Total	3	100%

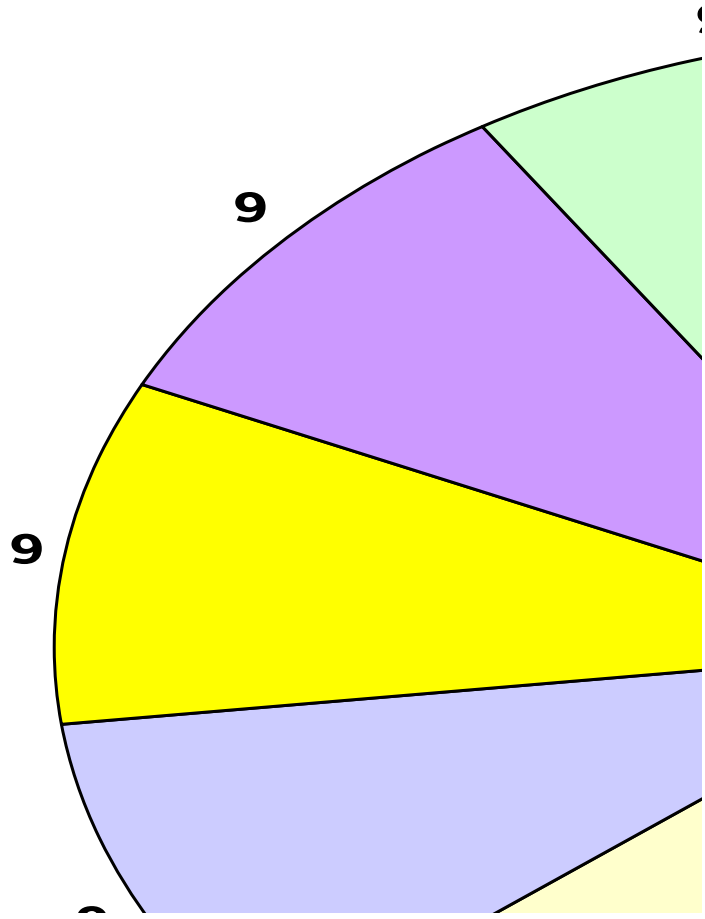
BIOPSY REPORT IN MALIGNANT ULCER



INFECTIVE ORGANISM IN DIABETIC ULCER

Organism	No.of Patients	Percentage
E.Coli	8	36.36%
Klebsiella	5	22.72%
Proteus	2	9.09%
Pseudomonas	2	9.09%
Staphylococcus	2	9.09%
Anaerobes	2	9.09%
Mixed	1	4.55%
Total	22	100%

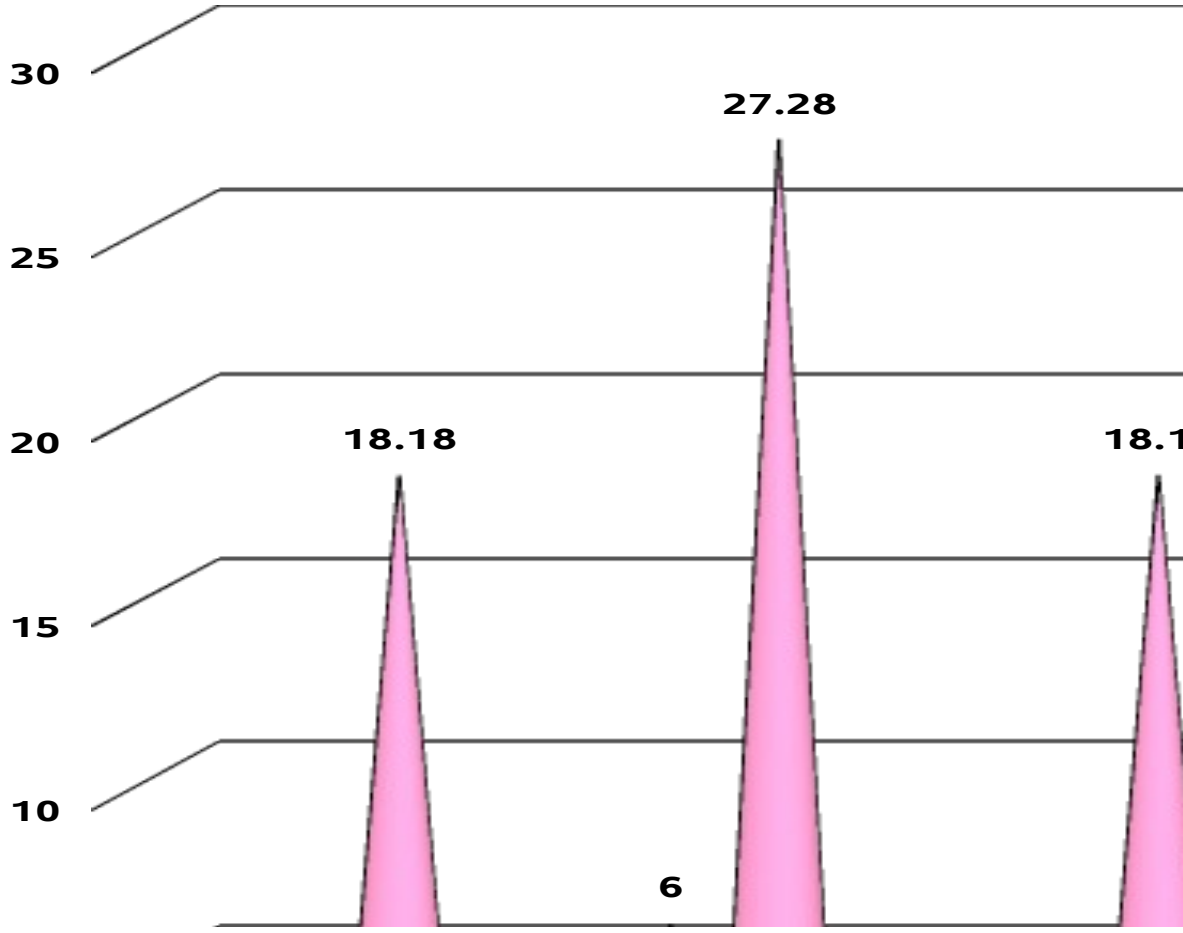
INFECTIVE ORGANISM IN DIABETIC ULCER



SENSITIVE DRUG IN DIABETIC ULCER

S. No.	Organism	No.of Patients	Percentage
1.	Cipro	4	18.18%
2.	Amikacin	6	27.28%
3.	Gentamicin	4	18.18%
4.	Pencillin	2	9.09%
5.	Cephalosporin	4	18.18%
6.	Metronidazole	2	9.09%
	Total	22	100%

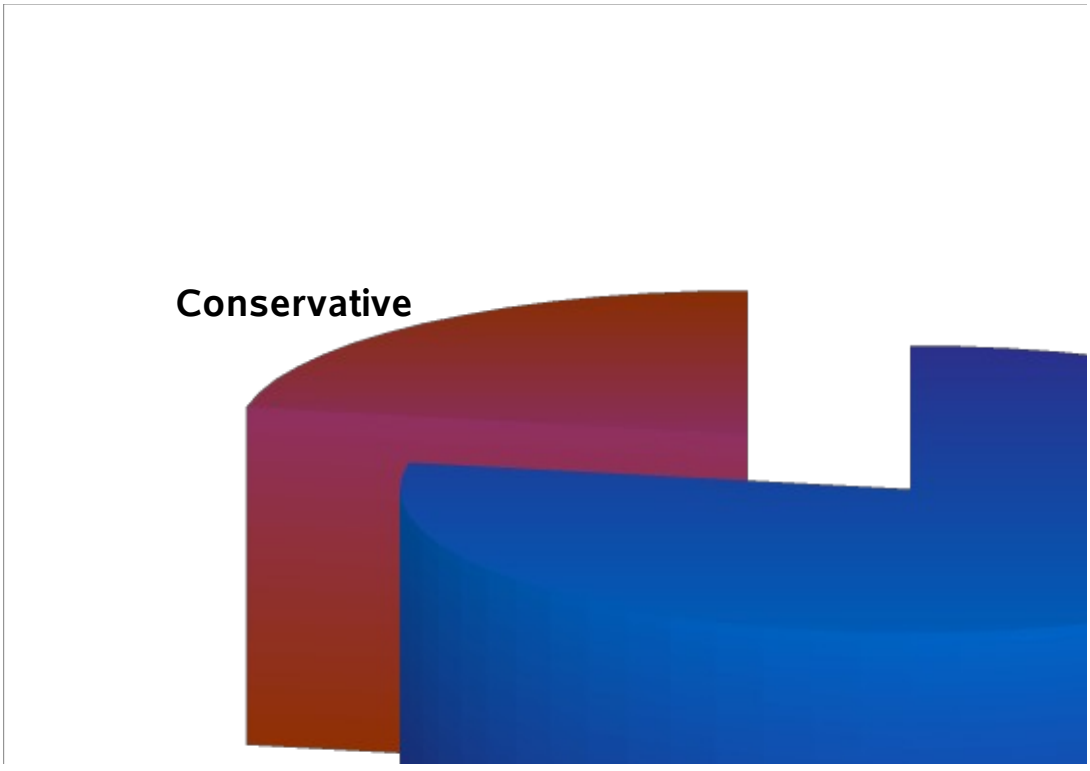
SENSITIVE DRUG IN DIABETIC ULCER



TYPE OF TREATMENT

S.No.	Type of Ulcer	No.of Patients	Treatment		Percentage	
			Conservative	Surgical	C	S
1.	Diabetic	22	4	18	8%	36%
2.	Arterial	17	2	15	4%	30%
3.	Malignant	3	-	3	-	6%
4.	Trophic	4	1	3	2%	1%
5.	Tuberculous	1	1	-	2%	-
6.	Others	3	3	-	6%	-
	Total	**50**	**11**	**39**	**22%**	**78%**

TYPE OF TREATMENT



INDICATION and INCIDENCE OF AMPUTATION

Type of Ulcer	Total No. of patients	No. of Amputee	% of Amputee
Diabetic	22	4	8%
Arterial	17	7	14%
Malignant	3	3	6%
Trophic	4	1	2%
Tuberculous	1	-	
Others	3	1	2%
Total	**50**	**16**	**32%**

LEVEL OF AMPUTATION

Level of amputation	No.of patients	%
Above elbow	2	12.5%
Below elbow	2	12.5%
Ray amputation	4	25%
Finger disarticulation	8	50%
Total	**16**	**100%**

RESULTS OF THE STUDY

RESULTS OF THE STUDY

- Epidemiology of this study revealed that patients with non-traumatic ulcers of upper limb constitute 0.2% of inpatients admission per year.
- Among the 50 cases studied diabetic ulcer constitute 44% crowns as the commonest cause of upper extremity ulcer in Govt. Rajaji Hospital.
- Ulcers due to vascular disorders and malignancy constitute the next common with 34% and 6% respectively.
- Commonest age group involved is above 40 years (76%) which correlated with other studies.
- In this study male patients occupy the predominantly affected sex with male : female 3:1.
- Arterial ulcers constitute about 30% are common in chronic smokers revealing that smoking is the commonest predisposing factor for arterial disorders.
- Pus culture and sensitivity done for diabetic patients reveal E.coli as the commonest organism 36% klebsiella 23%, Proteus 9%, when compared to standard studies, which mention mixed infections, staphylococcus and proteus as common organism.

- The sensitivity of organisms in diabetic ulcer studied shows Amikacin sensitivity in 27% and sensitive to ciprofloxacin, Gentamycin and cephalosporin in 18%.
- Most of the patients with upper limb ulcer need surgical treatment stressing on the importance of proper diagnosis and correct surgical procedure for better outcome.
- About 8% of diabetic ulcer patients underwent amputation, 14% of arterial ulcer patient underwent amputation either minor or major amputation.

CONCLUSION

CONCLUSION

Diabetic ulcer constitute the commonest cause of upper extremity ulcers (44%). Ulcers due to vascular disorders and malignancy coming next. Commonest age group involved is above 40 years. Arterials ulcers are common is smokers. Pus-culture and sensitivity done for diabetic ulcer patients reveled E-coli as the commonest organism and ciprofloxacin as the most sensitive drug. 32% of patients with upper extremity ulcer underwent amputation either minor or major amputation.

Prompt and proper diabetic control is essential. Efficient assessment clinics to evaluate the patients and select those who would benefit from intervention are essential to make best use of limited resources and also periodic review for prevention of recurrence and proper management.

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PROFORMA

PROFORMA

Name Age / Sex IP No.

Unit / Ward

History -*

Hand Ulceration duration

Site

Pain

Pigment and Itching

Bleeding

Discharge

Sensory Loss

Deformity

Past History

Diabetes Duration Treatment – Yes / No

Tuberculousis

Hypertension

IHD / Valvular heart disease

Personal H/o

Smoking

STD

Family H/o

- Congenital Hemolytic Anemia
- Connective Tissue Disorder

Examination

General

- Anemia
- Jaundice
- Xanthelesma
- Fever

Local

Inspection	palpation
Site	Temperature
Size	Tenderness
Shape	Edge
Margin	Base
Floor	Surrounding Area
Surrounding Area	Regional Node

Special Test

Diabetic and Trophic Ulcer – Sensory Testing Motor testing

Light touch	Power
Crude touch	Deformity
Joint sensation	

Vibration sensation

INVESTIGATION

Urine	:	Albumin		
		Sugar		
Blood	:	HB%	TC	DC
		Peripheral smear		ESR
Blood	:	Urea	Sugar	VDRL
Serum	:	Creatinine	Cholesterol	

PUS culture and Sensitivity - Organism

Sensitive Drug – Ci, G A, M, Ce, P

Biopsy Report of Ulcer :

X ray of local part Osteomyelitis present / Absent

Colour Doppler Study:

Final Diagnosis

Treatment

Type Conservative / Surgical

Amputation Yes / No

Level / Finger / AE / BE

Method guilliotine / Flap

Complication :

Follow up :

MASTER CHART

MASTER CHART

S. No.	Name	Age	Sex	IP No	Occupation	Diagnosis	P. Culture Organism	Drug	Treatment	Amputation	Complication	Follow up
1	Narayanan	38	M	26492	Agri	Diabetic	E.Coli	C	S			6
2	Subbiah	53	M	27852	Agri	Arterial			S	AE	WD	
3	Sathan	17	M	49904	Agri	Diabetic	Klebsiella	ceph	S			7
4	Siva subramani	54	M	26307	0FF	Trophic			S		WI	
5	Sathya	26	F	33540	H.W.	Arterial			C			15
6	Stephen	42	M	49468	Agri	Diabetic	Proteus	A	S	FD		
7	Sanglimuthu	33	M	27002	Agri	Arterial			S			
8	Mariammal	48	F	30106	Agri	Arterial			S	Ray		
9	Sakthivel	53	M	17945	Agri	Diabetic	E.coli	G	C			7
10	Krishnan	62	M	46448	-	Others			C			
11	Chinnammal	60	F	37246	Agri	Trophic			S		WI	
12	Narayanan	58	M	26311	-	Arterial			S			
13	Govindharaj	22	M	64211	Student	Diabetic	Klebsiella	A	C			8
14	Mookkammal	44	F	30207	-	Trophic			C			9
15	Selvaraj	50	M	61123	Agri	SCC			S	BE		15
16	Arumugam	52	M	42311	Agri	Diabetic	E.coli	C	S	FD		
17	Malliga	30	F	38121	H.W	Others			C			
18	Kardhavelan	60	M	38220	Agri	Arterial			S	Ray		6
19	Palani	62	M	28840	-	Diabetic	E.coli	A	C			
20	Ganesan	44	M	37246	Agri	Arterial			S		WI	7
21	Anbuselvi	48	F	46448	Agri	Diabetic	Pseudomonas	M	S	AE	B	6
22	Veerasamy	41	M	41133	Agri	Arterial			S	FD		
23	Anandh kumar	32	M	33171	Agri	Diabetic	Mixed	Ceph	S			
24	Seetharaman	52	M	30111	Agri	Diabetic						
25	Pondy`	58	M	37152	Agri	Diabetic						
26	Bala Subramani	50	M	4717	OFF	Diabetic	Anaerobe	A	C			6

S. No.	Name	Age	Sex	IP No	Occupation	Diagnosis	P. Culture Organism	Drug	Treatment	Amputation	Complication	Follow up
				3								
27	Selvam	54	M	33432	-	Diabetic	E.coli	Ceph	S	ED	WI	7
28	Srinivasan	64	M	34195	Agri	Arterial	Kelbsiella	G	S			10
29	Saravanan	44	M	33100	Agri	Arterial			S			
30	Saraswathy	42	F	21966	H.W.	Diabetic	Pseudomonas	A	S	Ray		
31	Ramanujam	38	M	19788	Agri	Arterial	E.coli	Ceph	S			
32	Chinna thambi	41	M	38512	-	Diabetic			C			4
33	Palanikumar	47	M	29400	Agri	Arterial	Klebsiella	C	S			
34	Periyasamy	43	M	17441	Agri	Diabetic			S	Ray		8
35	Murugan	53	M	20406	Agri	Trophic			S		WI	
36	Kamaraj	55	M	48541	Agri	Diabetic	Anaerobe	A	S			
37	Vairamani	60	F	17422	Cook	Malig.melanoma			S	FD		19
38	Subramani	62	M	33062	-	Tuberculous			C			8
39	Muthiah	37	M	6624	-	Diabetic	Staph	A	S			10

S. No.	Name	Age	Sex	IP No	Occupation	Diagnosis	P. Culture Organism	Drug	Treatment	Amputation	Complication	Follow up
				4								
40	Antony	64	M	47133	Agri	Arterial			S			12
41	Thangavelu	68	M	46780	Agri	Diabetic	E.coli	C	S			
42	Kasturi	50	F	37106	Agri	Arterial			S			
43	Rajamani	47	M	39732	Agri	Diabetic	Klebsiella	G	S	FD		7
44	Kulasekaran	46	M	32700	Agri	SCC			S	BE	WI	
45	Rasammal	18	F	47642	H.W.	Arterial			S			
46	Pandian	44	M	47176	-	Diabetic			S			9
47	Radhakrishnan	46	M	41236	Agri	Others			C			10
48	Ravishankar	52	M	41240	Agri	Diabetic	Staph	P	S			
49	Siva kumar	35	M	45506	Agri	Arterial			S		WI	7
50	Ponnalagu	50	F	47242	Agri	Diabetic	E.coli	M	S	FD		12

ABBREVIATIONS

ABBREVATIONS USED IN THE MASTER CHART

A	Amikacin	PL	Phantom limb
Agri	Agricultural labourer	Prot	Proteus
AE	Above Elbow	Pseud	Pseudomonas
BE	Below Elbow	Ray	Ray amputation
C	Conservative	R	Revision
Ce	Cephalosporin	S	Surgical
Ci	Ciprofloxacin	SCC	Squamous cell carcinoma
E.coli	Escherichia coli	Staph	Staphlococcus
F	Flap	TB	Tuberculous
G	Gentamicin	FD	Finger disarticulation
H.W.	House wife	Tech	Technical Job
Kleb	Klebsiella	Tt	Treatment
M	Metronidazole	WD	Wound dehiscence
Mm	Malignant melanoma	WI	Wound infection
Off	Officer	WE	Wide excision.
P	Pencillin		